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Cultural versus Biological Inheritance: Phenotypic Transmission from Parents to Children (A Theory of the Effect of Parental Phenotypes on Children's Phenotypes)

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INTRODUCTION

The foundations of biometrical genetics were first laid in a famous paper by R. A. Fisher [1]. Since that time a considerable amount of literature has accumulated, the book by Mather and Jinks [2] being the most recent summary. In most treatments, the mode of transmission of nonbiological contributions to the phenotype has not been specified in detail. An interesting exception is an analysis of adoption studies and IQ by S. Wright in 1931 [3], in which a direct inheritance of "environmental factors" over 1 generation has been postulated and found (by path coefficient analysis) to be of some magnitude.

When the parents' phenotypes affect the phenotype of their children directly, a new mode of inheritance arises which operates side by side with strictly biological inheritance (through the DNA) and may be difficult to distinguish from it. This transmission is entirely phenotypic. Under certain conditions its importance relative to purely biological inheritance is not negligible, and justifies its theoretical investigation. Such a mode of inheritance bypasses DNA and will be more relevant for those characters which develop through a learning process. Parents' participation in teaching is especially important in the earlier years of development, perhaps more so in the less developed areas of the world, and those traits that are learned during the first period of life are more likely to show phenotypic transmission from parent to child.

Clearly, effects of this kind are more likely to exist for traits where "cultural" transmission is of importance. In general terms, cultural transmission occurs when parents and other members of the group may influence a child's behavior. A preliminary analysis of the consequences of cultural effects of members of the group,

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as well as parents, has been given by Cavalli-Sforza and Feldman [4] and Feldman and Cavalli-Sforza [5]. In the present paper, however, we will limit our analysis to contrasting cultural and biological transmission from parent to child, omitting the effects of other members of the group. We shall also exclude the effects of natural selection and random genetic drift, assuming for simplicity that gene frequencies remain constant over generations. We shall study the effects that cultural transmission, occurring simultaneously with the established rules of biological inheritance, can be expected to have on the statistical measures commonly used for the analysis of biological inheritance. The main hope of separating the two different modes of transmission is through the study of adopted offspring, and we therefore shall also evaluate the expected effects of adoption.

THE MODEL AND DEFINITIONS

The basic assumption is that the phenotype of a child is determined by the phenotypes of his parents *and* the child's genotype. The phenotype of an individual will then be a function of his father's and mother's phenotypes, ϕ_F, ϕ_M, the function being determined by the individual's genotype i:

$$\phi_{im} = f_i(\phi_F, \phi_M) + \epsilon, \tag{1}$$

where ϵ is a random variate indicating random variation, with expected mean zero and variance σ^2. In what follows, ϕ_{im} is taken at generation t, and parental phenotypes at $t - 1$. The function f indicates the phenotypic response of an individual to that part of the environment which is formed by the phenotypes of the two parents of the individual. What is genotypic and therefore transmitted biologically is the mode of response to that environment, summarized by f.

The suffix i refers throughout to one of the genotypes in existence for each of which the function, f_i, must be specified and

$$\sum^{i}$$

will indicate summation with respect to all these genotypes. Matings for all different contributions of parental genotypes are to be considered separately and the suffix m will refer to one of such matings,

$$\sum^{m}$$

indicating summation with respect to all matings.

The probability of a given mating, m, will be called p_m; $p_{i\cdot m}$ is the probability of observing offspring genotype i from mating m (from standard Mendelian proportions) and p_i is the probability of genotype i in the population, so that

$$p_i = \sum^{m} p_m p_{i\cdot m}.$$

The conditional probability that a child comes from mating m given its genotype i is

$$p_{m\cdot i} = p_{i\cdot m}p_m / \sum^{m} p_{i\cdot m}p_m = p_{i\cdot m}p_m / p_i, \tag{2}$$

so that

$$\sum^{m} p_{m\cdot i} = 1. \tag{2a}$$

It will be assumed here that environmental factors other than parental phenotypes determine a random variation of individual values of ϕ_{im} with constant variance, σ^2, which may be taken to represent all other environmental factors. For some specific purposes, for example, evaluation of effects of order of birth or of social stratification, this restriction might be removed.

Denote by $\mu_i^{(t)}$ the *expected* value of the phenotype of individuals of genotype i at the tth generation. Then $\mu^{(t)}$, the *expected value of the mean of the whole population* at that generation will be

$$\mu^{(t)} = \sum^{i} p_i \mu_i^{(t)} = \sum^{m} p_m \sum^{i} p_{i\cdot m} E(\phi_{im}). \tag{3}$$

Expectations of the main quantities of interest are given below, all at time t.

The *total variance between individuals*

$$V_T^{(t)} = \sum^{m} p_m \sum^{i} p_{i\cdot m} E(\phi_{im} - \mu^{(t)})^2, \tag{4}$$

for ϕ_{im} at generation t, can be split into two additive components:

$$V_T^{(t)} = \sum\sum^{mi} p_m p_{i\cdot m} E(\phi_{im} - \mu_i^{(t)})^2 + \sum\sum^{mi} p_m p_{i\cdot m} (\mu_i^{(t)} - \mu^{(t)})^2. \tag{5}$$

The second term can be rewritten considering equations (2) and (2a):

$$\sum\sum^{mi} p_m p_{i\cdot m} (\mu_i^{(t)} - \mu^{(t)})^2 = \sum^{i} p_i (\mu_i^{(t)} - \mu^{(t)})^2 = V_G, \tag{6}$$

so that V_G is the weighted variance between expected means of genotypes. The quantity V_G is a natural candidate for a definition of *genetic* (genotypic) *variance*.

The first term can similarly be rewritten,

$$\sum\sum^{mi} p_m p_{i\cdot m} E(\phi_{im} - \mu_i^{(t)})^2 = \sum^{i} p_i \sum^{m} p_{mi} E(\phi_{im} - \mu_i^{(t)})^2, \tag{7}$$

and is the weighted expected *variance within genotypes,* and we may write

$$V_{ii}^{(t)} = \sum^{m} p_{mi} E(\phi_{im} - \mu_i^{(t)})^2 \tag{8}$$

as the variance within genotype i.

The *variance within families* is given by

$$V_W^{(t)} = \sum^{m} p_m E[\sum^{i} p_{i\cdot m} (\phi_{im} - \overline{\phi}_{im})^2], \tag{9}$$

when $\overline{\phi}_{im}$ is the mean phenotype of the offspring of a particular family of mating type m with an i offspring at generation t. The quantity V_W is also the variance between full sibs; $1 - V_W/V_T$ is the expected (intraclass) *correlation between sibs,* commonly denoted as r_{ss}.

To compute the *parent-offspring covariance* (not distinguishing between the

sexes of the parents) as the mean of the father-offspring and mother-offspring covariances, it is convenient to take the mean phenotype of the two parents (from the means of the $[t-1]$st generation),

$$\phi_m = \frac{\phi_F + \phi_M}{2}, \tag{10}$$

and correlate it with their children's phenotypes, ϕ_{im}:

$$W_{PO}{}^{(t)} = \sum^{m} p_m \sum^{i} p_{i \cdot m} E(\phi_m \phi_{im}) - \mu^{(t)} \mu^{(t-1)}, \tag{11}$$

where $\mu^{(t-1)}$, the parental mean, can be written $\mu^{(t-1)} = \sum^{m} p_m \phi_m$.
All four covariances (father-son, father-daughter, mother-son, mother-daughter) may be computed separately with a simple extension of formula (11) when the trait shows sex differences.

In addition, we are interested in the *covariance between adoptive parents and adopted offspring*:*

$$W_{FO}{}^{(t)} = \sum^{m} p_m \sum^{i} p_i E[\phi_m(\phi_{im} - \mu_A{}^{(t)})], \tag{12}$$

assuming adoption takes place at random, where $\mu_A{}^{(t)}$, the expected mean of the adopted offspring at time t, is

$$\mu_F{}^{(t)} = \sum^{m} p_m \sum^{i} p_i E(\phi_{im}). \tag{13}$$

We also want the *covariance between biological parents and their offspring which have been randomly adopted into other families*:*

$$W_{PF}{}^{(t)} = \sum^{m} p_m \sum^{m'} p_{m'} \sum^{i} p_{i \cdot m} E(\phi_m)\,[E(\phi_{im'}) - \mu_F{}^{(t)}]. \tag{14}$$

Here m refers to all matings (all combinations of parental genotypes) of the biological parents, m' to all matings of adoptive parents, ϕ_m to the mean parental phenotypes of the biological parents, and $\phi_{im'}$ to the phenotypes of the children, which are a function of their genotype i and of the phenotype of the adoptive parents.

One of the measurements which has been widely employed in human genetics to obtain an estimate of "broad heritability" is the *correlation between monozygous twins reared apart*. It is necessary to distinguish two categories of twins reared apart: (1) those pairs in which both twins have been reared in different adoptive families, AA; and (2) those of which one has been reared in an adoptive family and the other in the biological family, AB. The expected square differences between members of a twin pair, assuming adoptions are random with respect to genotype, are

$$D_{AA}{}^2 = \sum^{m} p_m \sum^{m'} p_{m'} \sum^{i} p_i E(\phi_{im} - \phi_{im'})^2, \tag{15}$$

$$D_{AB}{}^2 = \sum^{m} p_m \sum^{m'} p_{m'} \sum^{i} p_{i \cdot m} E(\phi_{im} - \phi_{im'})^2, \tag{15a}$$

* We use the notation F for "foster," referring in the first position to parent and in the second position to child.

where m, m' refers to all matings, those of the biological and the adoptive parents for AB, and the pairs of adoptive families for AA. Let j, k be the genotypes of the parents in the mth mating, and j', k' those of the m'th mating. Then,

$$\phi_m - \phi_{m'} = (x_j + x_k)/2 - (x_{j'} + x_{k'})/2, \tag{16}$$

where x_j, x_k are the phenotypes of independent individuals taken at random from the parental genotypes participating in mating m and $x_{j'}$, $x_{k'}$ are the same for mating m'. The expected values of equation (16) for insertion into equations (15) or (15a) can be computed given the means μ_j and the variances V_{jk}, and so forth, within each genotype, and knowledge of function (1).

The two intraclass correlations between monozygotic (MZ) twin pairs reared apart are

$$r_{AB} = 1 - D_{AB}^2/V_T \quad \text{and} \quad r_{AA} = 1 - D_{AA}^2/V_T. \tag{17}$$

The intraclass correlation between MZ twins not reared apart is

$$r_{MZ} = 1 - \sigma^2/V_T. \tag{17a}$$

The classical definition of "broad heritability" is

$$H = V_G/V_T. \tag{18}$$

Intraclass correlations between MZ twins reared apart may be expected to supply an approximate estimate of it. It is of considerable interest to determine the relationships between heritability defined in this way and the correlation above. We are in a position to examine a number of the approximations involved.

The above formulas are quite general and can be used numerically once function (1) is defined. They are valid for any number of genes or alleles and also for assortative mating. In the next section we explore analytically the simple case of one gene, two alleles, and random mating, when function (1) is linear in the mean of the parental phenotypes (see fig. 1).

THE CASE OF LINEAR DEPENDENCE ON PARENTAL PHENOTYPES

Consider the case of one pair of alleles A, a and a linear dependence on parental phenotype, so that the phenotype of the child, from equation (1), can be expressed as $\phi_{im} = a_i + 2b_i\phi_m + \epsilon$, where ϕ_m, the mean parental phenotype, is given by equation (10) and ϵ is a random variable with mean zero and variance σ^2 (see also fig. 1). The ϵ associated with different individual phenotypes are independent. Parameters a_i and b_i may differ in the three genotypes: $i = 1$ (AA); $i = 2$ (Aa); $i = 3$ (aa). Hardy-Weinberg equilibrium is assumed and p and q are the gene frequencies of A and a.

If the b_i are all zero, this case reduces to the standard one, usually analyzed in classical biometrical genetics, in which there is strict determination of phenotype from genotype alone, apart from random environmental variation.

The means of the three genotypes are given by

$$\underset{\sim}{\mu}^{(t)} = \underset{\sim}{a} + B\underset{\sim}{\mu}^{(t-1)}, \tag{19}$$

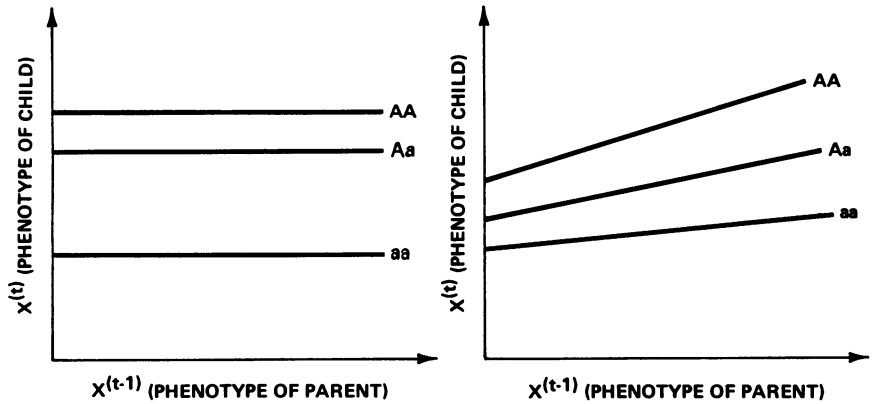

FIG. 1.—A one-gene, two-allele model with linear phenotypic dependence. *Left,* The classical genetic model: the phenotypic value of a genotype is independent of the parental phenotype. Here genotype *Aa* is represented as slightly dominant. *Right,* The phenotype of the child is a linear function of the parental phenotype, the function being different for every genotype. $X =$ phenotype, $t =$ generation.

where $\underset{\sim}{\mu} = (\mu_1, \mu_2, \mu_3)$, $\underset{\sim}{a} = a_1, a_2, a_3)$, and

$$B = \begin{bmatrix} 2b_1p & 2b_1q & 0 \\ b_2p & b_2 & b_2q \\ 0 & 2b_3p & 2b_3q \end{bmatrix}. \tag{20}$$

In the limit, independently of initial x_i values,

$$\underset{\sim}{\mu_i}^{(\infty)} = (I - B)^{-1}\underset{\sim}{a}^T \tag{21}$$

if

$$\sum_n B^n$$

converges. Here T stands for transpose. This condition is satisfied if all the eigenvalues of B are less than one in absolute value.

Simple formulas can be given when $p = q = 1/2$. Then,

$$(I - B)^{-1} = \frac{1}{\Delta}\begin{bmatrix} (1 - b_2)(1 - b_3) - b_2b_3/2, & b_1(1 - b_3), & b_1b_2/2 \\ b_2(1 - b_3)/2, & (1 - b_1)(1 - b_3), & b_2(1 - b_1)/2 \\ b_2b_3/2, & b_3(1 - b_1), & (1 - b_1)(1 - b_2) - b_1b_2/2 \end{bmatrix} \tag{22}$$

where $\Delta = 1 - b_1 - b_2 - b_3 + b_1b_3 + b_2(b_1 + b_3)/2$.

Always for $p = q$, one eigenvalue of the B matrix is zero, and the other two are

$$\lambda_{i,j} = \frac{b_1 + b_2 + b_3}{2} \pm \tfrac{1}{2}\sqrt{(b_1 - b_3)^2 + b_2^2}, \tag{23}$$

from which the condition of convergence can be easily tested. If the b are all equal,

convergence is assured if the b values are less than .5 in absolute value. Note that if, for example, $b_3 = 0$, then convergence may occur for one of b_1 or b_2 larger than .5.

The total variance at time t can be written from equation (4) as

$$V_T^{(t)} = \sum_{mi}\sum p_m p_{i\cdot m}[a_i^2 + 4a_i b_i E(\phi_m) + 4b_i^2 E(\phi_m^2)] - (\mu^{(t)})^2, \qquad (24)$$

where $E(\phi_m)$ is the expected parental phenotype computed from the appropriate values of $\mu_j^{(t-1)}$ and $\mu_{j'}^{(t-1)}$, and j and j' are the genotypes of the two parents in the six possible types of matings as in the third column of table 1. The $E(\phi_m^2)$ is the expected square of the mean parental phenotype and is given in general by

$$E(\phi_m^2) = (V_{jj} + V_{j'j'} + 2V_{jj'} + \mu_j^2 + \mu_{j'}^2 + 2\mu_j\mu_j)/4 + \sigma^2;\ j \neq j' \qquad (25)$$

$$E(\phi_m^2) = V_{jj}/2 + \mu_j^2 + \sigma^2;\ j = j',$$

where all V and μ refer to generation $t - 1$ (see also table 1).

TABLE 1

MATINGS AND EXPECTED VALUES OF MEAN AND MEAN-SQUARE PARENTAL PHENOTYPES (ϕ_m)

Mating m	Genotype of Parents	$E(\phi_m)$	$E(\phi_m^2)$
1	1, 1	μ_1	$V_{11}/2 + \mu_1^2 + \sigma^2$
2	1, 2	$(\mu_1 + \mu_2)/2$	$(V_{11} + 2V_{12} + V_{22} + \mu_1^2 + 2\mu_1\mu_2 + \mu_2^2)/4 + \sigma^2$
3	1, 3	$(\mu_1 + \mu_3)/2$	$(V_{11} + 2V_{13} + V_{33} + \mu_1^2 + 2\mu_1\mu_3 + \mu_3^2)/4 + \sigma^2$
4	2, 2	μ_2	$V_{22}/2 + \mu_2^2 + \sigma^2$
5	2, 3	$(\mu_2 + \mu_3)/2$	$(V_{22} + 2V_{23} + V_{33} + \mu_2^2 + 2\mu_2\mu_3 + \mu_3^2)/4 + \sigma^2$
6	3, 3	μ_3	$V_{33}/2 + \mu_3^2 + \sigma^2$

NOTE.—Genotype $1 = AA$, $2 = Aa$, $3 = aa$.

The μ_i are computed from equation (19) and the V_{ii}, forming the matrix of variances of phenotypic value within genotypes, can be computed by the following system of recurrence equations, where V' refers to generation t, and V, μ to generation $(t - 1)$:

$$V'_{11} = 2b_1^2[pV_{11} + 2pqV_{12} + qV_{22} + pq(\mu_1 - \mu_2)^2] + \sigma^2, \qquad (26)$$

$$V'_{22} = b_2^2[pV_{11} + qV_{33} + 2p^2V_{12} + 2pqV_{13} + V_{22} + 2q^2V_{23} + pq(\mu_1 - \mu_2)^2 + pq(\mu_2 - \mu_3)^2] + \sigma^2,$$

$$V'_{33} = 2b_3^2[pV_{22} + 2pqV_{23} + qV_{33} + pq(\mu_2 - \mu_3)^2] + \sigma^2.$$

Covariances for random pairs of individuals of the three genotypes are required in the general formulas above. They iterate independently from the variances as follows:

$$V'_{12} = 2b_1b_2[p(1+q)V_{12} + pqV_{13} + q^2V_{23}],$$
$$V'_{13} = 4b_1b_3[p^2V_{12} + pqV_{13} + q^2V_{23}], \tag{27}$$
$$V'_{23} = 2b_2b_3[p^2V_{12} + pqV_{13} + q(1+p)V_{23}].$$

The powers of the matrix of coefficients in this system converge to zero if all b are less than .5 in absolute value; and therefore the covariances tend to zero, irrespective of the initial values, if the μ_i values converge.

Let $\underset{\sim}{D}$ denote a column vector with terms

$$D_1 = 2b_1^2pq(\mu_1 - \mu_2)^2 + \sigma^2$$
$$D_2 = b_2^2pq[(\mu_1 - \mu_2)^2 + (\mu_2 - \mu_3)^2] + \sigma^2 \tag{28}$$
$$D_3 = 2b_3^2pq(\mu_2 - \mu_3)^2 + \sigma^2.$$

Then the equilibrium values of V_{ii} can be obtained from

$$V_{ii} = [(I - B^*)^{-1}\underset{\sim}{D}]_i, \tag{29}$$

where B^* is

$$B^* = \begin{bmatrix} 2b_1^2p & 2b_1^2q & 0 \\ b_2^2p & b_2^2 & b_2^2q \\ 0 & 2b_3^2p & 2b^3q \end{bmatrix}. \tag{30}$$

Conditions for convergence of the variances are satisfied by those of convergence of the means [see equations (21) and (24)]. The inverse of $(I - B^*)$ is easily obtained from that of $(I - B)$ [see equation (22)], squaring each b term in it.

The *total variance at equilibrium* can also be written from equations (5), (6), and (7) using the equilibrium values for μ_i, V_{ii} given by equations (21) and (31) as

$$V_T = p^2V_{11} + 2pqV_{22} + q^2V_{33} + V_G, \tag{31}$$

where V_G is the *genetic variance*

$$V_G = p^2\mu_1^2 + 2pq\mu_2^2 + q^2\mu_3^2 - \mu^2. \tag{32}$$

The *variance within families* iterates as follows:

$$\begin{aligned} V_W^{(t)} = {} & \sigma^2 + p^3q[(a_1 - a_2)^2 + 4(a_1 - a_2)(b_1 - b_2)E(\phi_2) \\ & + 4(b_1 - b_2)^2E(\phi_2^2)] \\ & + \frac{p^2q^2}{4}\{(a_1 - 2a_2 + a_3)^2 + 2(a_1 - a_3)^2 \\ & + 4E(\phi_4)[(a_1 - 2a_2 + a_3)(b_1 - 2b_2 + b_3) \\ & + 2(a_1 - a_3)(b_1 - b_3)] + 4E(\phi_4)^2[(b_1 - 2b_2 + b_3)^2 \\ & + 2(b_1 - b_3)^2]\} \\ & + pq^3[(a_2 - a_3)^2 + 4(a_2 - a_3)(b_2 - b_3)E(\phi_5) \\ & + 4(b_2 - b_3)^2E(\phi_5^2)], \end{aligned} \tag{33}$$

where the μ and V used in the calculations of $E(\phi_m)$, $E(\phi_m{}^2)$ (see table 1) must be from generation $(t-1)$. Alternatively, if the equilibrium values are used, V_W thus computed is the equilibrium value.

The *parent-offspring covariance* is

$$\begin{aligned} W_{PO}{}^{(t)} &= \sum_m^{} p_m \sum_i^{} p_{i\cdot m} E\{\phi_m[(a_i + 2b_i\phi_m) - E\sum_m^{} p_m \sum_i^{} p_{i\cdot m}(a_i + 2b_i\phi_m)]\} \\ &= [\sum_m^{} p_m E(\phi_m) \sum_i^{} p_{i\cdot m} a_i - \bar{a}\mu^{(t-1)}] + 2[\sum_m^{} p_m E(\phi_m{}^2) \sum_i^{} p_{i\cdot m} b_i \qquad (34) \\ &\quad - \mu^{(t-1)} \sum_m^{} p_m \sum_i^{} p_{i\cdot m} b_i E(\phi_m)], \end{aligned}$$

where

$$\bar{a} = \Sigma p_i a_i,$$

and for random mating as here,

$$\bar{a} = p^2 a_i + 2pqa_2 + q^2 a_3; \quad \bar{b} = \sum_i^{} p_i b_i = p^2 b_1 + 2pqb_2 + q^2 b_3. \qquad (35)$$

For analysis of foster children one must also consider their mean $(\mu_F{}^{(t)})$ and total variance, V_F. Then

$$\mu_F{}^{(t)} = \bar{a} + 2\bar{b}\,\mu^{(t)} \qquad (36)$$

and

$$\begin{aligned} V_F{}^{(t)} &= \sum_m^{} p_m \sum_i^{} p_i E(a_i + 2b_i\phi_m)^2 - \mu_F{}^{(t)} \qquad (37) \\ &= V_a + 4\mu V_{ab} + 4[\sum_m^{} p_m E(\phi_m{}^2) \sum_i^{} p_i b_i{}^2 - \bar{b}^2\mu^2], \end{aligned}$$

when V_a, V_{ab} are the variance of the a values and the covariance of the a, b values, respectively, weighted by the genotype frequencies.

The *covariances of adoptive offspring* are of importance since they offer the best hope of separately estimating functions of a and b values. The covariance of a foster parent with offspring is

$$W_{FO}{}^{(t)} = \bar{b}V_T{}^{(t)}, \qquad (38)$$

on the assumption that the covariances V_{12}, V_{13}, V_{23} of random pairs of genotypes are zero, as is true at equilibrium [equation (27)].

The *covariance of adopted offspring with the biological parent* is

$$\begin{aligned} W_{PF}{}^{(t)} &= [\sum_m^{} p_m E(\phi_m) \sum_i^{} p_{i\cdot m} a_i - \bar{a}\mu^{(t-1)}] \\ &\quad + [\sum_m^{} p_m E(\phi_m) \sum_i^{} p_{i\cdot m} b_i - \bar{b}\mu^{(t-1)}] 2\mu^{(t-1)}. \qquad (39) \end{aligned}$$

If the b are all equal, the latter term in square brackets vanishes: the overall result, however, will differ when $b_i = b$ from the biological parent-offspring covariance W_{PO} by

$$W_{PO}{}^{(t)} - W_{PF}{}^{(t)} = b[\sum_m^{} p_m E(\phi_m{}^2) - (\mu^{(t-1)})^2], \qquad (40)$$

which is zero only if b is zero. If $b_i = b$, then for $p = q = 1/2$, W_{PF} is given at equilibrium by

$$W_{PF} = (a_1 - a_3)^2/16(1 - b). \tag{41}$$

Formulas (15), (16), and (17) for identical twins can be used directly, computing $E(\phi_{im} - \phi_{im'})^2$ for equation (15) with j, j', k, k' defined as for equation (16) as

$$\begin{aligned} E(\phi_{im}^{(t)} - \phi_{im'}^{(t)})^2 = b_i^2\{&V_{jj} + V_{j'j'} + V_{kk} + V_{k'k'} + 2[V_{jj'}(1 - \delta_{jj'}) \\ &- V_{jk}(1 - \delta_{jk}) - V_{jk'}(1 - \delta_{jk'}) - V_{j'k}(1 - \delta_{j'k}) \\ &- V_{j'k'}(1 - \delta_{j'k'}) + V_{kk'}(1 - \delta_{kk'})] + \mu_j^2 \\ &+ \mu_{j'}^2 + \mu_k^2 + \mu_{k'}^2 + 2(\mu_j\mu_{j'} - \mu_j\mu_k - \mu_j\mu_{k'} \\ &- \mu_{j'}\mu_k - \mu_{j'}\mu_{k'} + \mu_k\mu_{k'})\}, \end{aligned} \tag{42}$$

where all V and μ refer to generation $(t - 1)$ and δ is Kronecker's delta.

The values of r_{MZAA} and r_{MZAB} are equal when all b are equal. In that case, the difference between H, the broad heritability, and r_{MZAA} (or r_{MZAB}) is, at equilibrium,

$$b^2(\mu_1 - \mu_3)^2/8V_T. \tag{43}$$

The implications of this difference are of some interest and are discussed in the next section.

NUMERICAL EXAMPLES

We will limit our numerical examples to the case of linear dependence, which has been treated algebraically in full. It is more likely that the true relationships, f_i [see equation (1)], are curved, but a linear approximation will usually be valid, at least in a restricted range. In principle, nonlinear dependencies can also be handled numerically by the general procedure given earlier.

Table 2 shows a collection of computations subdivided as follows: model B refers to genetic variation in the a values, the intercepts of the linear dependence between children's phenotype and parental phenotype. The b values are zero and therefore this is the classical model of biometrical genetics, to which our model reduces in that case. This is pure biological inheritance without any "plasticity," that is, the children's phenotype has no tendency to be molded by the parental phenotype a (quality expressed by the b values).

Model C refers to "pure cultural inheritance" without any genetic variation, in which the intercepts are all equal, and the b values, that is, the slopes of the linear dependence, are also all equal, but different from zero. It is clear that the hope of distinguishing between models of type B and C lies in adopted progeny. Correlations and regressions between adoptive parent and progeny are zero in B models, and in C models they are identical to those between the biological parent and offspring. Correlations between MZ twins reared apart are identical to the heritability in B models, and zero in C models.

TABLE 2

NUMERICAL VALUES OF THE MEANS, VARIANCES, CORRELATIONS, REGRESSIONS, AND HERITABILITY IN A SAMPLE OF CASES BELONGING TO FIVE TYPES OF MODELS

MODEL	INPUT PARAMETERS							GENOTYPE MEANS			GENERAL POPULATION AND FOSTER CHILDREN (F)			
											Means		Variances	
	a_1	a_2	a_3	b_1	b_2	b_3	σ^2	μ_1	μ_2	μ_3	μ	μ_F	V_T	V_F
B	2	1	0	0	0	0	0	2	1	0	1	1	0.5	0.5
	2	1	0	0	0	0	1	2	1	0	1	1	1.5	1.5
	2	2	0	0	0	0	0	2	2	0	1.5	1.5	0.75	0.75
	2	2	0	0	0	0	1	2	2	0	1.5	1.5	1.75	1.75
C	1	1	1	0	0	0	1	1	1	1	1	1	1	1
	1	1	1	.1	.1	.1	1	1.25	1.25	1.25	1.25	1.25	1.02	1.02
	1	1	1	.2	.2	.2	1	1.67	1.67	1.67	1.67	1.67	1.09	1.09
	1	1	1	.4	.4	.4	1	5	5	5	5	5	1.47	1.47
	1	1	1	.5	.5	.5	1	diverges						
PV	1	1	1	.5	0	−.5	1	3	1	0.33	1.33	1	2.52	2.52
	1	1	1	.4	.2	0	1	2.82	1.73	1	1.82	1.73	1.61	1.46
	1	1	1	.3	.2	.1	1	2.15	1.68	1.30	1.70	1.68	1.20	1.17
BC	2	1	0	.1	.1	.1	1	2.36	1.25	0.14	1.25	1.25	1.64	1.53
	2	1	0	.2	.2	.2	1	2.92	1.67	0.42	1.67	1.67	1.90	1.65
	2	1	0	.4	.4	.4	1	6.67	5	3.33	5	5	3.19	2.52
	2	2	0	.2	.2	.2	1	3.25	3	0.75	2.5	2.5	2.17	1.92
BCPV	2	1	0	.5	.25	0	1	6.4	2.4	0	2.8	2.4	7.47	5.28
	2	1	0	.5	.5	0	1	12	8	0	7.0	6.25	25.7	23.8
	2	1	0	.5	0	0	1	5	1	0	1.75	1.44	5.52	3.64
	2	1	0	0	.25	.5	1	2	2	2	2	2	1.26	1.24
	2	1	0	.5	0	−.5	1	5	1	−0.33	1.67	1	6.07	6.07
	2	1	0	0	−.25	−.5	1	2	0.62	−0.21	0.76	0.62	1.94	2.32
	2	1	0	−.5	−.5	0	1	1.18	0.47	0	0.53	0.60	1.89	2.00
	2	2	0	.5	.25	0	1	8	4	0	4	3.5	10.3	7.68
	2	2	0	.5	.5	0	1	16	12	0	10	9	46.8	45.6
	2	2	0	.5	0	0	1	6	2	0	2.5	2.12	6.58	4.37
	2	2	0	0	0	.5	1	2	2	2	2	2	1.17	1.15
	2	2	0	.25	.25	.25	1	3.83	3.5	1.17	3	3	2.38	2.05

TABLE 2 (*Continued*)

Model	Parent-Offspring					Sib-Sib	MZ Twins			
	Correlations			Regressions			Reared Together	Reared Apart		
	r_{PO}	r_{FO}	r_{PF}	b_{FO}	b_{PF}	(r_{SS})	(r_{MZ})	r_{AA}	r_{AB}	H
B	.5	0	.5	0	.5	.5	1	1	1	1
	.167	0	.167	0	.167	.167	.333	.333	.333	.333
	.333	0	.333	0	.333	.417	1	1	1	1
	.143	0	.143	0	.143	.179	.429	.429	.429	.429
C	0	0	0	0		0	0	0	0	0
	.1	.1	0	.1	0	.020	.200	0	0	0
	.2	.2	0	.2	0	.080	.080	0	0	0
	.4	.4	0	.4	0	.320	.320	0	0	0
PV	.235	0	.176	0	.176	.381	.603	.353	.331	.397
	.316	.210	.108	.200	.103	.282	.377	.257	.250	.262
	.233	.203	.031	.200	.030	.136	.165	.075	.073	.076
BC	.269	.104	.175	.100	.169	.240	.392	.372	.372	.375
	.364	.215	.176	.200	.164	.343	.474	.394	.394	.411
	.531	.450	.147	.400	.131	.608	.686	.366	.366	.436
	.344	.213	.153	.200	.144	.339	.540	.460	.460	.474
BCPV	.543	.297	.306	.250	.257	.670	.866	.679	.650	.707
	.572	.390	.273	.375	.263	.723	.961	.586	.619	.739
	.434	.154	.261	.125	.212	.553	.819	.694	.646	.668
	.268	.252	0	.250	0	.175	.205	.018	.009	0
	.390	0	.293	0	.293	.538	.835	.585	.549	.659
	—.058	—.229	.179	—.250	.196	.227	.485	.298	.291	.323
	—.332	—.365	.056	—.375	.057	.330	.471	.096	.089	.093
	.552	.289	.337	.250	.291	.672	.903	.715	.698	.777
	.552	.380	.260	.375	.256	.683	.979	.604	.644	.769
	.468	.153	.315	.125	.256	.566	.848	.723	.680	.722
	.143	.126	0	.125	0	.087	.143	.018	.009	0
	.390	.270	.151	.250	.140	.396	.580	.455	.455	.478

NOTE.—Model B = pure biological inheritance, zero plasticity; C = pure cultural inheritance, no difference between genotypes; BC = plasticity equal for all genotypes, but differences in intercepts; PV = genetic variations in plasticity only; BCPV = genetic variation both in intercepts and in plasticity. One gene–two allele model; gene frequency $p = .5$. The phenotype of children is a linear function of the mean phenotype of parents, the a values being the intercepts and the b values (plasticity) the slopes. Subscripts 1, 2, and 3 refer to the three genotypes AA, Aa, and aa; σ^2 is the environmental variation; heritability H is the variance between expected means of genotypes. All values at equilibrium.

In the third group, called model PV, there is genetic variation only in the b values (plasticity) and not in the intercepts a. The pattern thus created is similar, but not identical to that of genetic variation in the a values as in model B. Genetic variation in plasticity raises the correlations of MZ twins reared apart above zero but not as highly as in the B model.

The fourth group of values, model BC, refers to a situation of mixed biological and cultural inheritance, in which there is genetic variation in the intercepts and plasticity different from zero but equal for the three genotypes.

The fifth group, model BCPV, includes also plasticity variation of genetic origin. Clearly these models generate a great variety of patterns. In general, however, correlations between adoptive offspring and their foster parents tend to be less than those of biological parent and offspring but may be higher than for model PV. Correlations between MZ twins reared apart tend to be highest. The latter, as shown in equation (43), are similar but not identical to the broad heritability estimate and differ from it by a quantity which is a function of the b value.

There are eight independent parameters in the model, including the gene frequency (which was always taken equal to .5 in tables 1 and 2). When measurements of the trait are available, but the genotypes cannot be distinguished, there is still a chance, in principle, of fitting the model since there are more than eight statistics that can vary independently, at least to some extent: μ, μ_F, V_T, V_F, W_{PO}, W_{PF}, W_{SS}, r_{AA}, and r_{AB}. The fitting problem, however, is not easy; estimation depends heavily on adoption data and the model assumes random adoption, which is hardly the case. Some parameters (for example, regressions) are likely to be less affected by nonrandomness than, say, the mean M_F or the variance V_F, a possibility open to theoretical analysis. Further quantities not considered here might be introduced, such as the correlations between biological and adopted sibs.

Table 3 is built using a and b values that roughly fit IQ data. There are important limitations to using this model in its present form for analysis of IQ data, which will be considered in the discussion; for these reasons no attempt at a close fit was made but some simple results are presented for the sake of numerical illustration. The IQ data are standardized by a linear transformation to have mean 100 and variance 225; these restrictions were therefore imposed on the estimation, except in the last line of table 3 where the variance is higher. It should be noted that standardization of the variance of IQ involves a loss of information, and statistics of the untransformed variate or knowledge of the transformation employed, often not available in publications, would be useful. The estimate of broad heritability from separated MZ twins, averaged over the four existing studies, is about .8 [6]. The first line gives the expectation for mean 100, variance 225, and heritability .8 with the "pure biological inheritance" or standard model (model B of table 1). The comparison with observed correlations [7] invites the following considerations. The parent-offspring, sib-sib correlations are close to .5, as are also the observed values. Note, however, that assortative mating is known to be high for IQ, but is not considered in the computation of the theoretical correlations above, so that this agreement may be misleading. The B model leads to expectations of foster parent–child

TABLE 3

NUMERICAL VALUES OF THE MEANS, VARIANCES, CORRELATIONS, REGRESSIONS, AND HERITABILITY IN A SAMPLE OF CASES BELONGING TO FIVE TYPES OF MODELS, CHOOSING a, b, AND σ^2 VALUES THAT APPROXIMATELY FIT IQ MEAN, VARIANCE, AND HERITABILITY

MODEL	INPUT PARAMETERS							GENOTYPE MEANS			GENERAL POPULATION AND FOSTER CHILDREN (F)			
											Means		Variances	
	a_1	a_2	a_3	b_1	b_2	b_3	σ^2	μ_1	μ_2	μ_3	μ	μ_F	V_T	V_F
B	119	100	81	0	0	0	45	119	100	81	100	100	225	225
C	75	60	45	.2	.2	.2	40	119	100	81	100	100	227	171
PV	85	70	55	.15	.15	.15	60	118	100	82	100	100	222	182
BC	75	70	65	.20	.15	.1	60	119	100	83	101	100	224	184
BCPV	75	70	65	.28	.14	0	100	142	98	65	101	98	891	706

MODEL	PARENT-OFFSPRING					SIB-SIB (r_{SS})	MZ TWINS			H
	Correlations			Regressions			Reared Together (r_{MZ})	Reared Apart		
	r_{PO}	r_{FO}	r_{PF}	b_{FO}	b_{PF}			r_{AA}	r_{AB}	
B	.400	0	.400	0	.400	.400	.800	.800	.800	.800
C	.510	.231	.357	.200	.310	.576	.824	.744	.744	.775
PV	.448	.166	.329	.150	.298	.477	.730	.685	.685	.701
BC	.449	.166	.328	.150	.298	.480	.732	.685	.684	.701
BCPV	.511	.157	.406	.140	.361	.579	.888	.829	.822	.848

NOTE.—No attempt at a close fit was made for reasons given in text. See table 2 for explanation of symbols.

correlations (r_{FO}) of zero; the observed values are in the range of .2–.4. Thus, a plasticity $\bar{b}$ [see equations (35) and (38)] different from zero is indicated. The observed values, however, may have to be reduced if they need correction for "matching" (see Discussion). The lines after the first indicate models with average plasticity $\bar{b}$ between .14 and .2, with and without genetic control of plasticity.

DISCUSSION

There is a wide gap today between the thinking of extreme environmentalists, usually from sociological or psychological circles, who do not concede to the importance of genetic determination of behavioral traits, and of those who do not concede to the importance of the environment of upbringing and its variation in the determination of individual behavior. Some of the differences that exist between these lines of thought are brought into focus by the model we have developed and are thus given a better chance of objective estimation and analysis. Our model subsumes a variety of simpler ones, extending from that espoused by the extreme environmentalists, to that assuming complete genetic control. Between the two extremes is a continuum of intermediate models. Variety is generated by the relative role of individual "plasticity." This is at its maximum in the environmentally oriented models, and at its minimum at the other extreme. Plasticity refers to the capacity to respond to the environment. We have concentrated in this paper on the effects of the environment that is formed by the parents of the developing individual, and plasticity defined here is the capacity to respond to that fraction of the environment. One reason for concentrating on the parental environment is its presumed importance in shaping certain traits. Another is that the methods of analysis of inheritance rely heavily on the correlations between relatives. Parent-offspring interaction of the kinds envisaged here affect not only parent-offspring correlations but also many others (sib-sib, etc.). The analysis of the observed correlations between relatives forms the basis for understanding the mechanism of transmission. The neglect of plasticity in such analyses may seriously undermine the validity of conclusions, and this may be one cause of resistance to accept the evidence for biological inheritance derived from correlations between relatives. We will see that some of this resistance is justified. In a vein similar to the present one, the analysis might be extended to encompass the effects of sib-sib interactions and others. Interactions with members of the group other than relatives have already formed the basis of an analysis of rates and consequences of cultural evolution [4, 5].

Especially for behavioral traits in mammals and other vertebrates, learning processes contribute heavily to the development of behavior. It would therefore seem that with such traits a consideration of plasticity is essential. Plasticity can itself be controlled by genes which may or may not be the same as those which determine the "baseline" of a trait being studied. In the model we have envisaged, and, in particular, the linear version which we have explored in more detail, the baseline of a trait is roughly expressed by the a constants (the intercepts), and the plasticity by the b constants (the slopes) of the straight lines expressing the

phenotypic response of the child to the parental phenotypes. Naturally, baselines have a meaning only in relation to the origin and scale on which the trait is measured. Plasticity is scale-independent, but its numerical value may be affected by the actual range of values of the trait being investigated, when the functional relationship between the trait in a child and that in the parents is not linear over the full range.

One result of importance is the prediction that, given the existence of individual plasticity in response to the environment, correlations between biological relatives are expected *even if there is no genetic variation whatsoever.* We have called this "pure cultural transmission" on the assumption that it will be of special importance in some behavioral and cultural traits. But for physical traits a similar situation may arise, for instance, when cultural transmission affects the diet and this affects a physical trait. Thus correlations can be created between biological relatives for physical traits even in the absence of genetic variation, but they arise indirectly, being mediated by the existence of cultural variation and inheritance of customs or preferred environments. For behavioral traits, on the other hand, the effects of cultural variation and inheritance can be direct, and a phenotype-to-phenotype transmission may arise which parallels but should be kept distinct from that of biological inheritance through DNA. Model C of table 2 refers to this pure cultural (or phenotypic) transmission, which corresponds to the posture of extreme environmentalism.

At the other extreme, model B refers to pure biological inheritance with zero plasticity. This coincides with the standard model of biometrical genetics in which the attempt is sometimes made to take account of environmental effects by splitting constants accounting for environmental variation into various components, for example, between families (E_2) and within families (E_1) [2], and genotype-environment interactions. While such a treatment may be rigorous for experiments in plants and animals, in man it fails to meet the fact that the peculiarities of a family environment are, in part, transmitted culturally to descendants as is explicitly postulated in our model. It also cannot substitute for a direct investigation of the role of individual plasticity.

The distinction between a pure B-type and a pure C-type model can be made without difficulty by comparing correlations of adoptive relatives and biological ones. In C-type models, adoptive relatives correlate between themselves just as much as biological relatives do, while in B-type models adoptive correlations are zero and only biological ones are positive. However, when both types of transmission operate, a complex confounding takes place. Complexity is even greater on recognition that genetic variation may be present not only at the level of the base value taken by a trait, when uninfluenced by parental environment (as expressed by our a intercepts), but also at the level of individual plasticity (our b values). This is likely to be the case for many behavioral traits. A cornerstone of the analysis is the correlation or, preferably, the regression of adopted children to an adoptive parent. We have seen that this measures the mean plasticity $\bar{b}$. A value of zero in this correlation does not, incidentally, rule out entirely a genetic variation in plasticity,

for there might be genotypes with positive and genotypes with negative b values, having an average close to zero. This is perhaps only a theoretical possibility at this stage. More serious is the objection that correlations between adoptive parents and offspring such as have been found, for instance, for IQ, might be created by the attempts of adoption agencies to "match" the biological and the adopting families. Such matching, if it really is extensive and effective, would certainly obscure the value of adoptive correlations for estimating plasticity, unless its effect can be removed by partial regression. A further caveat is that the estimate of plasticity by regression of a trait in adopted children on the *same* trait in their adoptive parents, as investigated here, is not a comprehensive measurement of plasticity for the trait (even if limited to the response to the parental environment). The IQ in children may be to some extent molded by parental traits other than their IQ [8]; a consideration of general plasticity of a trait should take these "unspecific" contributions into account. A more general model than the present one would therefore be necessary for taking them into account.

It is of interest that adoption will also affect means and variances of adoptive children, which may differ from those of the general population. Unfortunately, means and variances of adopted children will be largely biased (probably more than regressions or correlations) by nonrandomness in adoption, making the interpretation of adoption data more difficult.

From tables 2 and 3 it can be seen that unless the b values are zero, correlations between MZ twins reared together in the biological families are always higher than the heritability H. The overestimate may be even worse if there are more environmental similarities between twins than between sibs; here there was no attempt to differentiate between the environmental variation of twins and nontwins.

Also of interest is the fact that correlations between MZ twins reared apart, which certainly play a major role in distinguishing between the various models, come close but do not exactly estimate broad heritability, defined as the ratio of variance between genotype means to total variance. The MZ twins reared apart in different adoptive families (AA) and those reared apart one in the biological family and the other in the adoptive family (AB) give different correlations under conditions that have been specified, but the difference between them is small. Sometimes, however, they give results that underestimate or overestimate broad heritability by more than 10%. If the b values are equal, MZ twins reared apart always underestimate heritability H [see equation (43)]; if the b values differ, however, they may overestimate it. One may add that H [see equations (6), (18), and (32)] cannot be directly estimated in practice unless individuals can be assigned to genotypes, and even then the expected variance between genotypes can be shown to be generally substantially larger than the genotypic variance V_G (the variance between expected means of genotypes) used for the computation of H.

These considerations are, of course, independent from other limitations of studies of MZ twins reared apart: the difficulties of evaluating the varied influences of a common life in utero (see also [6]); the delay in adoption after birth; the fact that usually MZ twins reared apart are examined at the same age, which varies greatly from pair to pair in the four studies available (see [6] for references). There seems

to be a tendency for the correlation to fall with age of the pair. Possible genetic effects on the pattern of IQ change with age are not clearly understood since longitudinal studies on twins are extremely limited and contradictory [9, 10]. Finally, limitations due to correlations between adopting and biological families have not been, perhaps, fully appreciated. In the largest study of MZ twins reared apart [11], limited to schoolchildren of London, minimal information on socioeconomic conditions of the biological and adopting family is given; but in the next largest study [12], which has been reported in a much more detailed way, the adopting family is in 61% of the cases closely related biologically (the child's grandparents or uncles and aunts).

Some of the numerical examples we have considered approximately fit the IQ data, but it would be premature to consider them more than numerical exercises. Here we have developed theoretically a one-gene, two-allele model (with random mating), while most of the IQ variation (except at the low end of the distribution) is polygenic. This limitation is actually not too serious, for most polygenic models are very simple extensions of one-gene, two-allele models. More important for a fully satisfactory analysis of IQ data is the extension of the model to include assortative mating and social stratifications. This will require further work.

Behavioral disorders, ranging from schizophrenia to alcoholism, may be treated by this model provided suitable data exist. Even if information is coded in the form of a threshold trait, the basic correlations can be obtained by the use of tetrachoric correlation functions [13].

These results stress that without the study of adoptions (of which MZ twins reared apart are an especially valuable but also a very rare example), correlations between relatives are not sufficient to discriminate, in full rigor, biological from cultural inheritance.

Another conclusion of general interest is that the processes of cultural inheritance, unlike the biological ones, do not reach equilibrium in 1 generation. All the formulas of means, variances, covariances, and derived statistics given in the text are valid for a given generation; changes in the overall environment, as when an ethnic group transfers to a new culture, involve changes in these statistics. The rate of approach to equilibrium depends largely on the eigenvalues of the B and the B^* matrices, being slower for greater b values. Several generations may be necessary if the initial means of the trait are widely different from those at equilibrium, as can be easily tested by iterating equation (19) (see fig. 2). This may be of special relevance, for instance, to IQ differences of ethnic groups transferred to new cultural environments or conditions. For cultural traits for which the b values are above .5, there may exist no upper limits to the means, which could then increase indefinitely.

SUMMARY

In the classical theory of biometrical genetics, the genotype determines the phenotype of an individual, the effect of the environment being expressed by one or more constants, but the interplay of genotype and environment is not recognized as a dynamic factor. The concept that the phenotype is the result of development of an individual in a certain environment and that the genotype provides the mode of

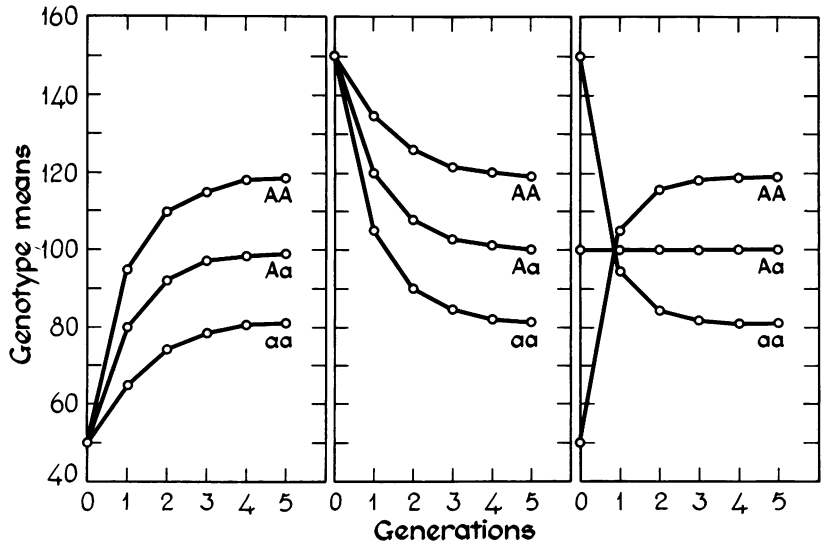

FIG. 2.—Change with time of the means of the 3 generations in a BC-type model, with $a_1 = 75$, $a_2 = 60$, $a_3 = 45$, $b_1 = b_2 = b_3 = 0.2$, $\sigma^2 = 40$, and $p = .5$, starting at generation 0 with three different sets of initial values of the genotype means.

reaction, but the environment provides the challenges and the conditions of development, is basic to genetic thinking; but in the classical models of biometrical genetics the representation of this interaction is usually absent or limited to the evaluation of genotypic-environment interaction variances.

In experimental situations, such as are possible with plants and animals, the classical model may usually suffice. The simplicity of the model is then still adequate because suitable experimental designs can help minimize or evaluate genotype-environment interactions. In nonexperimental situations, as are usual in man, this approach seems unsatisfactory especially for behavioral traits, where it is clear that the phenotype is the consequence of a long and complex learning process. A fraction of the teaching-learning process takes place through parent-offspring interaction and another fraction, which we have explored elsewhere, is affected through age-peer, or teacher-pupil interaction, or in still other ways. Side by side with biological transmission, a purely "cultural" inheritance thus arises, which in the case of parent-offspring interaction is almost completely confounded with biological inheritance. Correlations between relatives are affected by both cultural and biological inheritance, and the distinction of the contributions of the two mechanisms of transmission is not simple.

Formalization of the contribution of cultural transmission to a trait is possible, and we have investigated the consequences of parent-offspring cultural transmission. Our model also includes genetic variation of various kinds. It thus encompasses a variety of models, from one extreme of pure cultural transmission to the other of pure biological transmission. This last model is that of classical biometrical genetics. The major hope of analysis of real data comes from the parallel study of covariances

of adoptive and biological relatives. Even though this method has practical limitations, it is basic to a proper understanding of inheritance, especially for behavioral traits.

The expectations for the standard correlations and regressions have been computed here for a general model of phenotypic transmission for any type of dependence of the child's phenotype on that of the parents, and for a special simple case in which the child's phenotype is linearly related to that of the parents. The slope of this relationship expresses the average "plasticity" of the phenotype with respect to that fraction of the environment offered by the parents' phenotypes for the trait being examined. Some evidence is available that plasticity is not negligible, even for traits such as IQ. The consequences of plasticity have been explored and expectations obtained, in general and for specific examples. Some possibilities of obtaining estimates from an analysis of adopted offspring, including their meaning and validity, have been considered.

It is unfortunate that the available data are exceedingly sparse so that few firm statements can be made today for any specific trait. The conclusion seems inescapable, however, that only through careful studies of data on both adopted and biological offspring can the issue of cultural versus biological inheritance, and their relative roles, be settled in a rigorous way. The dynamic nature of all statistical values and, therefore, the necessity for some time to elapse before they reach equilibrium after environmental changes is also to be noted.

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